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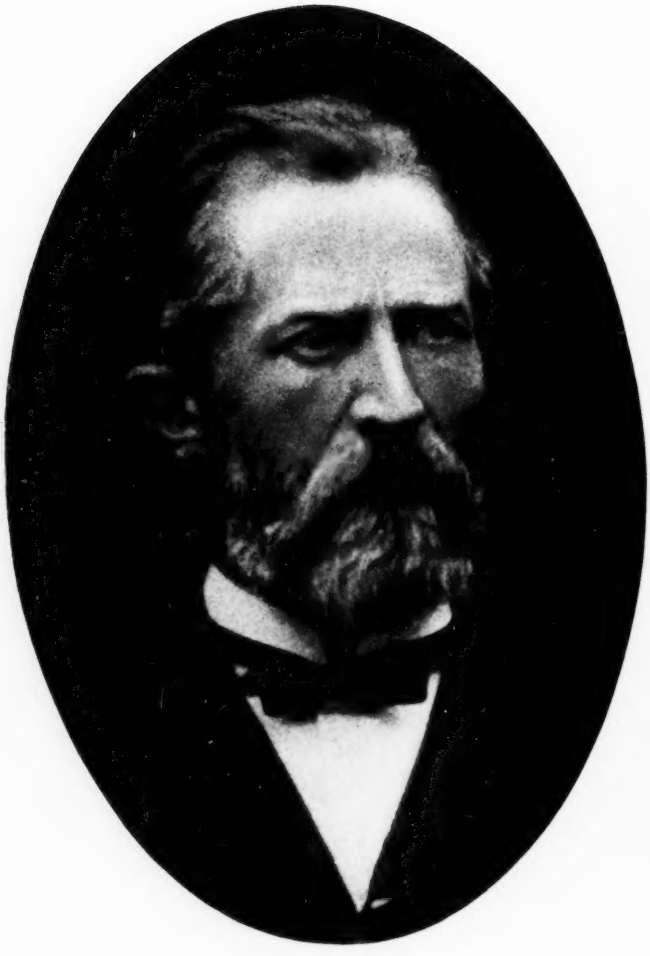

ANTON DE BARY

# PHYTOPATHOLOGY

VOLUME I NUMBER 1

FEBRUARY, 1911

## ANTON DE BARY

ERWIN F. SMITH

(WITH PORTRAIT, PLATE I.)

Of all the personalities contributing to the advancement of plant pathology from its crude beginnings to the present time, none has been more interesting than that of De Bary, none more productive of important results. It is fitting, therefore, that his portrait should appear in the first issue of a new journal of plant pathology, the first journal of its kind to be published in America. May it be indicative not only of the breadth and inclusiveness of the new journal, but also of its standard of excellence.

De Bary was born in January 1831 at Frankfurt am Main. He became interested in botany while he was yet a student in the Gymnasium. He graduated from the Gymnasium in 1848 and as Doctor of Medicine in Berlin in 1853. He practiced for a few months in Frankfurt and then abandoned medicine for research in botany. He began as privat docent in Tübingen in December, 1853. Two years later he became Nägeli's successor in Freiburg, first as ausserordentlich, and then (1859) as ordentlich professor, where he remained until 1867. That year he refused a call to Leipzig and accepted one to Halle in Schlechtendal's place. In 1872 he was called to the University of Strassburg, where he held the chair of botany until his death in January, 1888.

The photograph was taken in 1887, and represents him in the full maturity of his powers.

De Bary attracted to his laboratory students from all parts of the world, and through a long series of years, commencing in 1852, he contributed in rapid succession a series of monographs on algae, fungi, myxomycetes, bacteria, and higher plants, in which biological adaptations were always kept in mind, and in which profound thought rivaled clear expression and admirable delineation in forcing home conviction. At the same time this wonderful man found opportunity to write difficult books covering whole fields of knowledge, to edit the

*Botanische Zeitung*, the leading botanical journal of his time, to carry on correspondence, write reviews, give lectures, do administrative work, and keep everybody who came within his reach, by tongue or pen or printed page, stirred up and stimulated to do his best. Dying at 57 he left an impress on his generation distinctly peculiar. No less than 68 men afterwards distinguished in science studied under him at Strassburg. His work and that of his students put plant pathology on a new foundation, and he also, undoubtedly, had much influence on human and animal pathology, since his very successful infection experiments with fungi on plants suggested many things to those who were trying to determine the cause of human and animal plagues. DeBary's work cleared the way for all that has followed in plant pathology, and we must ever think and speak of him with that reverence due to a great master.

WASHINGTON, D. C.

# THE RUSTS OF WHITE AND RED CLOVER

FRANK D. KERN

Although it was pointed out by Liro[1] several years ago that the rust of white clover, *Trifolium repens*, is different both in structure and habit from the rust of red clover, *T. pratense*, most American mycologists seem either to ignore or to have overlooked the fact. On account of the economic importance of the clovers mention is often made of rust, along with other parasitic diseases, in catalogs, plant disease surveys, Experiment Station and other publications, but so far as the writer knows there is no suggestion in American literature that the white and red clover rusts may be distinct. For instance, if one turns to Duggar's Fungous Diseases of Plants,[2] one finds a heading, CLOVER RUST, *Uromyces Trifolii* (Hedw.) Lev.; and under the discussion of habitat relations is the following remark, "Among the clovers the following species are frequently infested: red clover (*Trifolium pratense*), hybrid clover (*T. hybridum*), white clover (*T. repens*), and crimson clover (*T. incarnatum*)." This clearly gives the idea that these four clovers are affected by the same rust-species which is transferrable from one to the other hosts, and yet Duggar evidently was aware of certain differences since he remarks that "the spermogonial and aecidial stages are not commonly found upon red clover, the host upon which the other stages are perhaps most frequent." He, however, apparently finds nothing else to arouse any suspicion and explains that the absence of the spermogonia and aecidia on red clover may, perhaps, be taken to indicate "that the teleutosporic form does not so readily infect this species of host."

The Sydows[3] in their Monograph of the genus *Uromyces* published in 1909, give rather full and accurate descriptions of the rusts themselves, together with some notes, but the writer cannot agree with them in their disposition of the names and their assignment of some of the hosts. In general they follow Liro in their treatment. The white clover rust is considered distinct; it has all four spore forms, pycnia, aecia, uredinia, and telia, and so far as they have observed does not occur on any other species of clover. According to them it differs from the red clover species in the possession of the aecial generation and in the smaller number of germ-pores in the urediniospores. Following Liro they take up as the name of this white clover rust *Uromyces Trifolii-repentis* (Cast.) Liro, giving *Aecidium Trifolii-*

[1] Liro, J. Ivar. [J. I. Lindroth], in Acta Soc. pro Fauna et Flora Fennica **29**: 15. 1906.

[2] Duggar, B. M., Fungous Diseases of Plants, 395. [1909.]

[3] Sydow, P. & H., Monographia Uredinearum **2**: 131–134. 1909.

*repentis* Cast.[1] as the only synonym. The name *Uromyces Trifolii* (Hedw. f.) Lev. is reserved for the red clover rust, which is said to be on six other species of clover, also.

The writer agrees to the separation of the white and red clover rusts into two species and finds practically the same morphological differences as suggested above. The species inhabiting white clover has 3–4 equatorial germ-pores while the species on the red clover has 4–6 scattered germ-pores. The white clover rust is beyond question an autoecious species commonly producing all spore forms in succession on the same host. This has been experimentally proved, also, by both European and American investigators.[2] There is still some doubt concerning the life-history of the red clover rust. The Sydows state that they have never seen aecia on red clover or on any of the other hosts considered to bear the same rust and this is also the experience of the writer. An aecial stage may possibly occur occasionally upon some of the other clovers aside from *T. repens*, but it is the opinion of the writer that in all such cases it is a part of and belongs with the uredinia and telia of the white clover rust and is not necessarily an indication that the red clover rust is an autoecious species. Rostrup claims to have collected aecia on *T. hybridum* and that seems not only possible but quite probable because an investigation of the other stages on *T. hybridum* show that they are similar to the white clover species. All of the specimens on *T. hybridum* examined by the writer, representing eight different states and one foreign country are clearly referable to the white clover species. Dietel reports aecia on *T. incarnatum* from Wisconsin and also from Italy. This, too, seems not improbable for an examination of the uredinial stage on *T. incarnatum* hosts shows that the 3–4-pored urediniospores also occur on this species of host. Judging from the specimens examined thus far by the writer the indications are that *T. incarnatum* may serve as a host for both of the rust species under discussion. To go a step further it is not impossible that red clover itself, *T. pratense*, may very rarely harbor the white clover rust. Howell[3] reports the detection of aecia on a plant of red clover and appends a note that so far as could be ascertained the aecial stage had never before been reported in America upon red clover, but that there seemed to be no doubt that the form was identical with that on white clover. In the artificial cultures Howell also produced uredinia on one red clover plant from infection with aeciospores (presumably taken from white clover although not so stated). Duggar

[1] Castagne, Louis, Observ. 1: 33. 1842.

[2] Howell, J. K., Bull. Cornell Univ. Agric. Exper. Station **24**: 129–139. 1890.

[3] Howell, J. K., l.c.

observes that the aecial stage commonly occurs upon *T. carolinianum* but that is known to belong to *Uromyces elegans* (B. & C.) Lagerh., a well defined species lacking the uredinial stage, and thus not coming into the present discussion.

In consideration of the foregoing the writer has come to the conclusion that evidence is entirely lacking to show that the rust, possessing urediniospores with 4–6 scattered pores and commonly occurring on red clover and allied species, is autoecious, producing aecia on the same hosts. On the contrary, there seems to be more justification for considering it heteroecious. In all the years in which rust has been known and collected on the clovers there is but a single uncertain report of an aecial stage on red clover and this, as has been explained above, doubtless belongs to the white clover rust. This constant failure of an aecial stage to be associated on the same leaves or plants with the uredinial and telial stages may usually be taken to indicate that, if it exists at all, it is to be looked for upon another set of host plants, i.e., it is likely to be heteroecious. The further fact that a number of other similar rusts of legumes are known to be heteroecious also lends support to this theory. Since a number of similar leguminous rusts are known to have their aecial phase on *Euphorbiaceous* hosts of the group to which *Euphorbia commutata* belongs, it seems not unlikely that the red clover rust may also have its aecia here.

Regarding the names of these two rusts of white and red clover, respectively, the writer must call in question the disposition of Liro and the Sydows. They employ the name *Uromyces Trifolii* (Hedw. f.) Lev. for the red clover species, but an examination of the Flora Francaise, vol. 2, p. 225, where the specific name *Trifolii* was originally published shows the fact very clearly that it, too, belongs to the white clover species. The name is proposed as follows :

604. Puccinia des trefles. *Puccinia Trifolii.*

*Puccinia Trifolii.* Hedw. f. Fung. ined. t. 18.

α. *Trifolii repentis.*
β. *Trifolii filiformis.*
γ. *Trifolii hybridi.*

Next is given a description and then the following sentence concerning distribution and hosts : "J'ai trouvé cette espèce dans un pré ombragé, près Fontenai-aux-Roses : elle croît sur le trefle rampant, le trefle filiforme et le trefle hybride."

It does not seem possible to interpret this in such a way as to make the name applicable to any other than the rust of the white and hybrid clover. A further examination of the names published for the rusts of clover does not reveal any one applicable to the red clover species prior to *Uredo fallens* Desm. of 1843. The only name listed in Sydow's

Monograph as belonging to the red clover species and published prior to 1843, which could possibly be tenable is *Uredo apiculata* Strauss in Ann. Wett. **2**: 97, 1811. This is published as *Uredo apiculata* α *Trifolii* and cites *Trifolium hybridum* as the chief host thereby making it clearly applicable to the other species.

*Uredo fallens* was published in the Plantes Cryptogames de France par J. B. H. J. Desmazieres, Fasc. 27, edit. 1, no. *1325* issued in 1843. He accompanies this with a brief but applicable description and says that it is found on the leaves of *Trifolium*. This is indefinite but an examination of the specimen shows at once that it is *Trifolium pratense* and a microscopical examination shows that the urediniospores have the characteristic scattered germ-pores. In an appended note Desmazieres remarks that this has up to this time been confused with *Uredo apiculosa* var. *Trifolii* from which it differs in certain particulars, mentioning the paler brown sori and the deeper color of the spore walls under the microscope.

The writer in view of these facts would, therefore, take up the name *fallens* as the specific name for the red clover rust and since it has never been transferred to the genus *Uromyces*, here proposes the new combination. The synomyms and hosts of the two rusts discussed in this paper are as follows:

**Uromyces fallens** (Desm.) nov. comb.

*Uredo fallens* Desm. Pl. Crypt. France [edit. 1] *1325*. 1843.

O & I. Pycnia and aecia unknown but presumably existing, probably on *Euphorbia*.

II & III. Uredinia and telia on

? *Trifolium incarnatum* L. (Crimson clover)
*Trifolium medium* L. (Zigzag clover)
*Trifolium pratense* L. (Red clover)

UROMYCES TRIFOLII (Hedw. f.) Lev. Ann. Sci. Nat. Bot. $8^3$: 371. 1847.

*Puccinia Trifolii* Hedw. f.; DC. Fl. Fr. **2**: 225. 1805.
*Uredo Fabae Trifolii* A. & S. Consp. fung. 127. 1805.
*Uredo Trifolii* DC. in Poiret, Encyl. meth. **8**: 223. 1808.
*Aecidium Trifolii-repentis* Cast. Obs. **1**: 33. 1842.
*Trichobasis fallens* Cooke. Micr. Fungi. ed. 2, 225. 1870.
*Uromyces Trifolii-repentis* Liro, Acta. Soc. Fauna et Fl. Fennica **29**: 15. 1906.

O, I, II & III. Pycnia, aecia, uredinia, and telia on

*Trifolium incarnatum* L. (Crimson clover)
*Trifolium hybridum* L. (Alsike clover)
*Trifolium repens* L. (White clover)

COLUMBIA UNIVERSITY,
NEW YORK CITY

# CROWN GALL OF PLANTS

ERWIN F. SMITH

(WITH PLATES II AND III)

This disease is characterized by marked overgrowth of the affected tissues. It occurs on plants of many families and in various regions of the world. In the United States it has been designated "crown gall" because it occurs very frequently on the crown of the plant, but it is found also on roots and on stems above the earth surface. In Europe where it occurs on grape, rose, raspberry, beet, etc., it is known by a variety of names: canker, krebs, wurzelkropf, broussins, rogna.

For the past six years team work has been done on crown gall in the Bureau of Plant Industry by the writer and three colleagues, Dr. C. O. Townsend (first five years), Miss Alice C. Haskins (first two years), and Miss Nellie A Brown (last four years). Other co-workers have also contributed a little here and there,—cultures set, sections cut, flagella stained, chemical analyses made.

A bulletin on this subject is in preparation and to this publication those readers are referred who desire further proofs in confirmation of the statements here affirmed.

## CAUSE OF THE DISEASE

This disease is due to *bacteria*: not to fungi, not to myxomycetes, not to mites, not to frosts, not to disturbances in nutrition. All these alleged causes have had their champions but they are not the true cause. They are all *post hoc* inferences based for the most part only on microscopic examinations made, in so far as organisms have been incriminated, in the later stages of the disease, when various saprophytes are certain to be present. Of clear cut and convincing experimental evidence in their favor there is not a scrap.

Bacteria were seen by the writer in fresh unstained sections of immature and non-necrosed crown galls in February, 1904, and two years later we obtained convincing proof of the gall-producing power of a particular schizomycete, obtained from galls by means of agar poured-plates, sub-cultured on agar from the colonies, and inoculated into sound plants. This organism was then named *Bacterium tumefaciens* S. & T. Since that date our experiments have been continuous, and we have obtained successful inoculations from pure cultures and sub-cultures on more than a thousand plants. Sometimes we have failed but gradually we have come to know many of the sources of our failures, and nothing in our experiments has led us to wish to modify in any material way our first published statement (Science, April 26, 1907, and Centralbl. f. Bakt., Abt. 2, Bd. XX, No. 1/3, 1907), or

subsequent statements (Science, Feb. 12, 1909 and August 13, 1909). As evidence in point the reader may consult the two plates accompanying this text, remembering that these results were obtained by pure culture inoculations, not by graftings (we have made no grafting experiments), and that we have obtained hundreds of others equally convincing; *e. g.* of 43 hops inoculated with pure cultures from the daisy (two experiments, one by Miss Brown and one by myself) 42 developed galls where punctured; of 85 sugar beets inoculated with pure cultures from the daisy (5 experiments) 83 contracted the disease, and 82 only at the point of inoculation; of 12 carnations inoculated by Townsend every one developed tumors. On the other hand with rare exceptions our numerous controls have remained free. We may then admit that these overgrowths are due to bacteria. Are they all due to the same organism?

## CROSS INOCULATIONS

Early in our experiments we began to make inoculations on a variety of plants. The first work was done on the gall of the Paris daisy, but as opportunity came we made platings from tumors on other plants. Up to the present time we have obtained tumor-producing schizomycetes from overgrowths on plants belonging to many widely separated families (Compositae to Salicaceae). These organisms are closely alike on various culture media, and many of them are readily cross-inoculable, *e. g. daisy* to peach, radish, grape, sugar-beet, hop; *peach* to daisy, apple, Pelargonium, sugar-beet, poplar; *hop* to daisy, tomato, sugar-beet; *grape* to almond, sugar-beet; *poplar* to cactus, oleander, sugar-beet; *willow* to daisy. Some cross-inoculate more readily than others, and there are also slight cultural differences. The morphological differences are trifling. In general it may be said that all plants susceptible to crown galls, *i. e.*, those on which they have been found in nature, are susceptible to artificial cross inoculation. The extent of natural cross-inoculation is less easily determined, but there is a good body of evidence going to show that it often takes place when one kind of plant follows another, *e. g.*, when peaches follow galled raspberries or apples.

## CULTURAL AND OTHER DIFFICULTIES

*Bacterium tumefaciens* dies readily in some of the common culture media, and also gradually loses its virulence. It often occurs in the tumors in a cultivable condition only in extremely small numbers so as to be missed very readily. It also comes up on agar plates poured from the galls more slowly than the saprophytes and the latter were the first colonies we considered, and also undoubtedly the ones which have

attracted the attention of other workers. Both yellow and white non-pathogenic schizomycetes occur in crown galls and colonies of some of the latter on agar closely resemble the tumor-producing organism. Furthermore the organism does not make pockets or abscess cavities in the tumors such as are common in many bacterial diseases of animals and plants. Moreover, it does not stain well in the tissues and consequently is certain to be overlooked in any ordinary examination of stained slides. The microscope alone cannot be depended upon. We should never have gotton anywhere had we depended solely or principally upon that instrument. The best proof of the existence of the organism in the tissues is our ability to obtain it (sometimes in nearly or quite pure culture) by the poured-plate method from young non-necrosed galls after thorough surface sterilization. Old galls are unsuited for preparation of cultures.

### HARD VS. SOFT GALLS

Various writers have maintained that there are two kinds of galls, especially on the apple tree, viz. hard galls and soft galls, the former being non-infectious and comprising most of the specimens met with. This has been a favorite argument of certain dealers in trees and Dr. Hedgcock at one time maintained this view. Our experiments, however, do not warrant any such distinction. The writer produced slow-growing hard galls on apple trees by inoculation with an organism taken from a soft gall of the peach; and the hardest of hard galls of apple, selected for us by Dr. Hedgcock as typical, yielded colonies closely resembling those plated from soft galls, and with some of these colonies galls were produced on other plants. Whatever may be the factors leading in the apple to hard gall in some cases and soft gall in others both are due to infectious bacteria, and both kinds of gall are to be avoided in making plantings. Owing to the slow growth of these galls on many apple trees it has been maintained with some show of reason that they are harmless, and in some cases this may be true, although in the photograph of two rows of apple trees one galled and the other not, submitted by Dr. Hedgcock in support of this contention, the writer was able immediately to pick out the galled row before he had seen the legend. But even if this contention were generally true, which it is not, there are other reasons why all galled trees should be rejected in planting, namely because the gall is not covered by a smooth, sound, *protective* bark layer, and consequently often affords a ready entrance to other parasites. Some convincing details are given in the bulletin.

### APPLE HAIRY ROOT

This disease supposed to be distinct from crown gall and non-infectious we have also proved to be due to bacteria scarcely distinguishable from those occurring in ordinary crown gall. These bacteria are found in the nodular base from which the tufted clusters of roots arise. We have induced the disease by pure culture inoculation not only in the apple but also in other plants and the disease would seem to belong with crown gall as only a special form of the latter. As a tentative hypothesis until further experiments are made we may assume either: (1) That the hairy root organism while resembling the crown gall organism is not identical with it; or (2) that they are the same, and if infection takes place in a certain group of cells an ordinary gall will develop, while if other special groups of cells are first invaded, *i. e.* root anlage, then a cluster of the fleshy roots will develop. Some of our inoculation experiments point to the latter conclusion.

### NATURE OF THE GALL

Superficially these growths are like those due to various fungi, to gall insects, to *Plasmodiophora brassicae*, or to the olive-tubercle organism, but structurally they are different. Their manner of growth and their histology strongly suggest certain malignant animal tumors. The writer has twice called attention to this publicly at meetings of the American Society for Cancer Research (first in 1909) and has summarized the evidence somewhat carefully in the bulletin referred to. More recently (October, 1910) Jensen of Denmark has expressed similar views respecting the crown gall of the sugar-beet, but without knowing of our results.

One of the principal biological problems of today, and one of the most interesting in the whole range of pathology, is to determine if possible the chemical stimulus responsible for overgrowths. This problem is of immense and wide-reaching importance not only to the pathologist but also to the physiologist and all others interested in cell mechanics. In conclusion I may be permitted to express the hope that in these plant tumors, now so easily producible by a definite microorganism, we possess means of determining the cause of cell division and possibly of shedding some light on the origin of certain malignant animal tumors.

NOTE.—Massee has recently claimed to confirm Toumey's finding of *Dendrophagus* in crown gall of rose and chrysanthemum (Kew Bulletin No. 9, 1910). He also notes that he could find no trace of *B. tumefaciens*. The last statement is not surprising, and the first one appears to rest on no better evidence than a somewhat hasty microscopic examination, without attempt at pure cultures or inoculations. Such a microscopic examination, even if it were a very long and careful

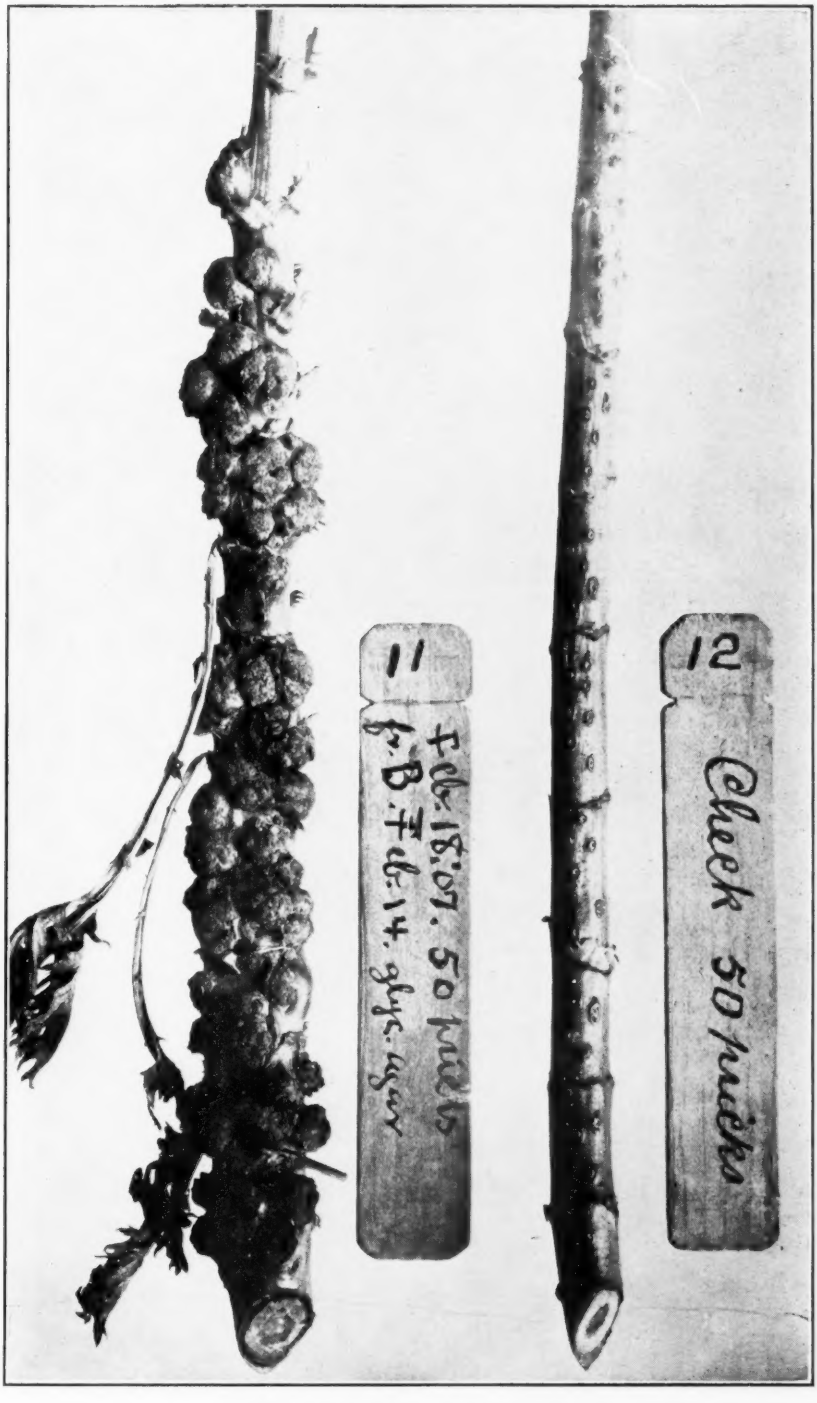


PLATE II. **Smith on Crown Gall.**

11. Fifty punctures with an infected needle; 12, fifty punctures with a sterile needle. The young inoculated plant forked into two twin branches of which 11 is one and 12 the other. Time two months and four days. Photo. April 22, 1907. Pure culture inoculation of daisy on daisy.

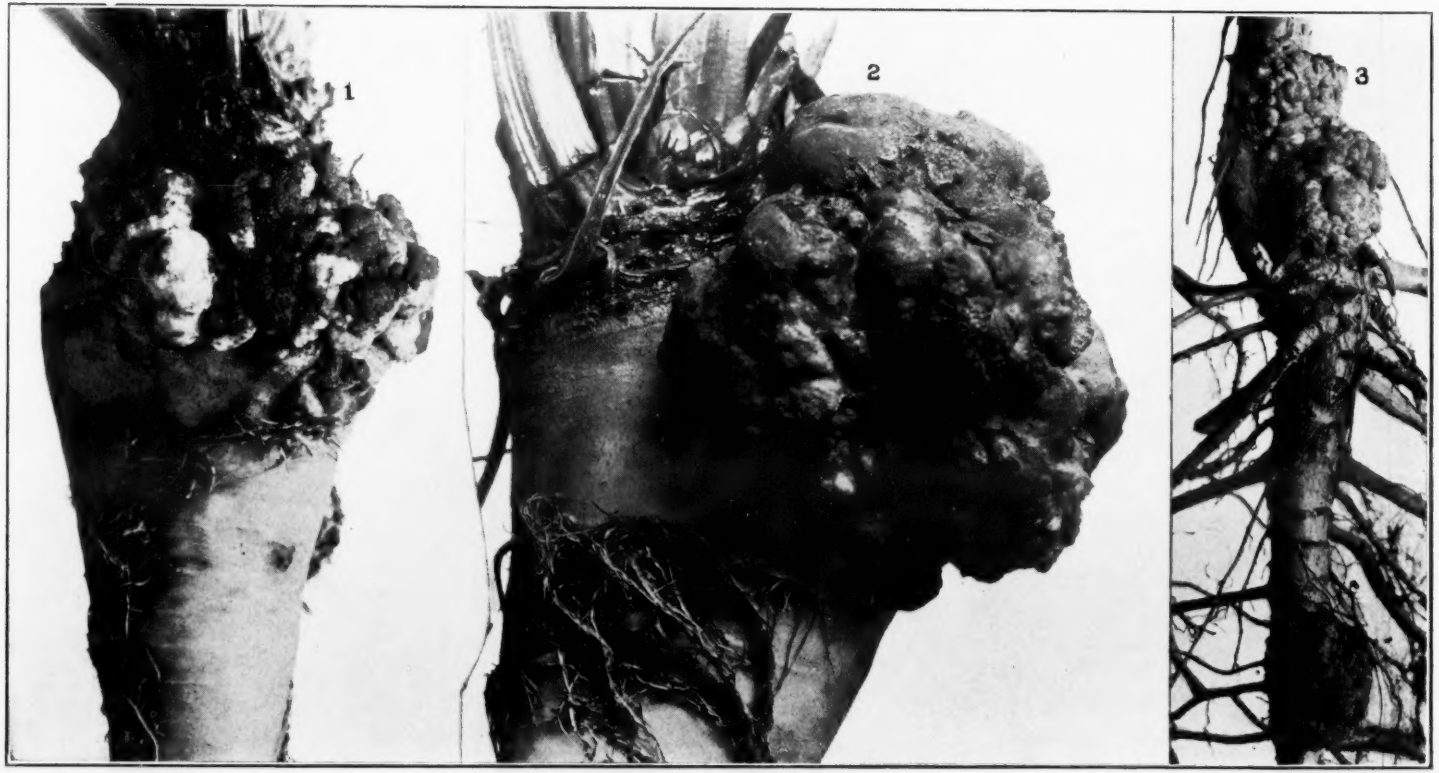


PLATE III. **Pure Culture Inoculations of Crown Gall.**

1. Grape on sugar beet, 47 days. Of 12 plants, 11 contracted the disease at place pricked. 1910.
2. Hop on sugar beet, 60 days. Inoc. in 1910 from about 26th sub-culture. Of 5 plants, all developed tumors.
3. Peach on peach, 50 days. Of 60 trees inoc., 55 developed tumors. The checks remained free. 1908.

one, would not enable any one to decide with certainty whether the granulations visible in certain cells should be regarded as the plasmodium of a myxomycete, or only as products of cell disorganization, which are often peculiarly liable to misinterpretation. The writer saw and studied Prof. Toumey's slides, but was never convinced that the organism described and figured by him (Arizona Bull. 33, page 54, figs. 28 and 29) had any connection whatsoever with the cell-changes in the gall also described and figured by him, *e. g.*, those on the plate facing page 49. But even granting Toumey's interpretation as correct, the etiological significance of the plasmodium has not been made out, since all sorts of non-parasitic soil organisms are likely to occur in galls when they are of some size. Toumey inoculated only ten plants altogether and obtained successful infections on *three* only of the ten. The inoculations were made in a country where the disease prevailed extensively and with spore material taken from the *cut surface of the gall*. The *Dendrophagus* spores, therefore, would be liable to contamination with anything occurring on the cut surface, especially the virulent schizomycete which can be demonstrated to be present in such galls and with which in pure sub-culture the disease can be reproduced, if the plants are in a young and actively growing condition, 100 times out of 100.

The writer has never seen anything on or in crown galls which has in any way led him to think that Myxomycetes have anything to do with their production. Phenomena similar to that described by Toumey were seen independently by the writer as long ago as 1893 in cells of the crown gall of the peach, and after six months' work upon the subject, the conclusion was reached that the appearances were artefacts and not living slime molds.

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# TWO NEW FIG DISEASES

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(WITH PLATE IV AND ONE FIGURE IN THE TEXT.)

The diseases to which the fig tree and its fruit are subject in the southern states seem to be very little known. The fig tree grows so easily and produces such an abundance of fruit that the loss due to the several diseases has been overlooked, or it has not seemed of sufficient importance for study. The fig tree is subject to seven or more diseases in the state of Louisiana. Of these, five are fairly well known, at least to botanists, while the other two are new. One of these new diseases, which I am calling the Fig Canker, is due to a new species of fungus; while the other, the Limb Blight, is due to a fungus which is not new but has not been known to occur on the fig tree.

## THE FIG CANKER

The fig canker has been under observation for three years. It is characterized by the shrinking and drying out of the tissue around a fruit scar, accompanied by an increased growth of the healthy surrounding tissue (Pl. I, Fig. 2), and followed later by a dropping out of the dead tissue thus leaving an open wound in the branch. The distribution of the disease does not seem to be widespread, as I have not noticed it in any region other than that in the vicinity of Baton Rouge, though I have searched for it in a number of localities in different parts of the state. The loss caused by the disease under present conditions is not great, on account of the abundance of fig trees and their rapidity of growth. However, if the time comes when more is made of the fig industry the disease may become of economic importance. The damage done by the disease is, 1st: the killing of many of the young branches, 2nd: the weakening of the branches in the neighborhood of the cankers thus allowing them to be more easily broken by the wind, and 3rd: the formation of open wounds in the branches which increase the chances for heart rots to develop.

The cankers are perfectly characterized at all stages in their development. At first, the tissue surrounding a fruit scar turns slightly darker in color and shrinks, often showing, also, a number of pink pustules on the surface. This diseased portion gradually spreads out in all directions until the branch may be from one half to two thirds girdled. Rarely is the girdling complete, because the twig dies before this stage is reached. When the twig is not more than half girdled, it generally is not killed, and so the scars of cankers may be seen on branches of all sizes.

In the healthy tissue surrounding the canker, generally there is an increased growth, which is in many respects similar to the healing over process which occurs in all woody plants. In many cases from a short distance away, the canker appears like a knot on the branch, due to this increased growth of the healthy tissue. But, though the tree tries to heal over the wound caused by the canker, it is never very successfully done, even after the causative organism is dead. This is due, I think, to the other saprophytic fungi and also insects which work on the dead tissue of the canker.

A longitudinal section of a cankered branch shows that the fungus which causes the disease has penetrated and killed the bark, cambium and part of the wood. This is readily told by the dark color which the diseased portions assume. The fungus threads are present in all the discolored parts with the exception of the pith, which is also often discolored.

As the canker grows older, the dead tissue cracks and finally, with the aid of scavenger beetles and other insects, falls out, leaving an open wound which sometimes extends nearly to the center of the branch. These large open wounds may be seen on branches which are two or three inches in diameter, several years after the fungus, which caused the disease originally, is dead.

Infection seems to take place in the fruit scar alone and within less than a year after the development of the fruit. But as the fungus grows slowly, the canker does not appear for several months after the infection. As the fruit is borne on the first year wood, the canker does not form until that portion of the twig is in its second year. The cankers develop all through the spring, summer, and fall, or from March to September.

The cause of the canker is a new species of fungus which I am calling *Tubercularia fici*. While the fungus does not fit in all respects the genus *Tubercularia*, it more nearly approaches that than other related genera. Material was placed in the hands of Professor G. F. Atkinson of Cornell University who kindly examined it. He also believed that it would be better to place the fungus in this genus than to establish a new one. It is probably the imperfect stage of some ascomycete, and its disposition at the present can at the best, be but a temporary one.

The fungus forms cushions of pseudoparenchymatous tissue on the surface of the cankers (Fig. 1, a), these sometimes beginning their development underneath some of the layers of cells of the host and finally breaking through, and sometimes forming directly on the surface. These cushions are very irregular in shape and size. Sometimes

the surface is rounded and smooth, while in other cases, it is very irregular. From these cushions of fungus tissue, the small narrow conidiophores develop. The small, elliptical, hyaline spores (Fig. 1, c) are cut off one at a time from the ends of the conidiophores. At the time the spores are developing, the pustules are a light pink in color.

The one character possessed by this fungus which is most divergent from the typical *Tuberculariа*, is the presence of peculiar setae (Figs. 1, a–b) which are scattered throughout the sporodochia. The setae are quite abundant, are very long and threadlike, hyaline or nearly so, and are papillose or echinulate. No genus, so far as we were able to determine, has such peculiar setae.

The fungus was readily cultured, both from spores and from pieces of the diseased tissue. The fungus grows rather slowly in pure culture, usually forming a perfectly white growth. Spores may or may not develop in the cultures. Some cultures did not produce any spores at all, some produced them freely on the aerial mycelium, while one culture produced them in pink pustules on the surface of the medium. But with all cultures, the ability to produce spores on culture media diminished after a few transfers. The setae were also produced scatteringly in some cultures.

To prove that this fungus was the cause of the cankers, some inoculations were made on March 9, and March 22, 1910. The inoculations were made by inserting some of the mycelium or spores from a pure culture into a small abrasion made on the fruit scar with a knife. Part of the inoculations were covered with grafting wax while the rest were left unprotected. For the checks, the fruit scars were slightly wounded with a knife. Three cultures were used in the inoculation as follows :

No. 1245. The fungus was cultured in December, 1909, from material collected during the preceding fall. This culture was characterized by the presence of pink pustules of spores.

No. 1247. Fresh cankers were found on some fig trees on March 9, 1910, and the spores were immediately cultured. Spores formed abundantly on the aerial mycelium in the culture giving the mycelium a very faint pinkish tint, but no pustules were found.

No. 1276. The culture was made in November, 1909, by transferring pieces of the diseased tissue of the cankers to tubes. The spore formation in this culture was very scanty.

The infection took place slowly, similarly to the way it takes place naturally. The cankers first began to show in June and from then on until August when the twigs were cut and carefully examined. It is probable that a slightly higher per cent of infection would have been

obtained, if the twigs had been left on the trees for a month or so longer. As the inoculations were made in the Station garden, where many people were allowed to pick the figs, a few of the tags marking the inoculations were lost, but not enough to vitiate the results.

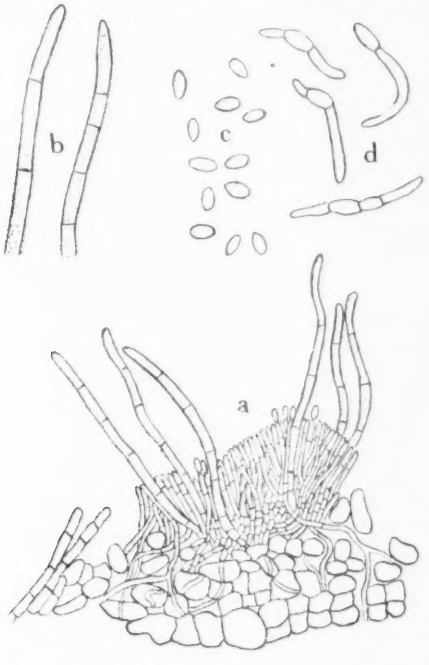


Fig. 1. *Tubercularia fici*. *a*. Sketch of sporodochium, showing setae, conidiophores, and spore formation. × about 350. *b*. Portions of setae, showing characteristic markings. × 750. *c*. Spores. × 750. *d*. Germination of spores in potato agar, after 18 hours. × 750.

The results of the inoculations are given in the following table. The inoculations that were protected with grafting wax are marked "wax", while those not so protected are marked "no wax".

RESULTS OF INOCULATIONS.

| Inoculations | Date | Number of Inoculations | Number lost | Infected | Doubtful | No infection |
|---|---|---|---|---|---|---|
| Check, wax | Mar. 9 | 5 | 0 | 0 | 0 | 5 |
| Check, no wax | Mar. 9 | 5 | 1 | 0 | 0 | 4 |
| 1245, wax | Mar. 9 | 14 | 1 | 7 | 3 | 3 |
| 1245, no wax | Mar. 9 | 16 | 0 | 0 | 5 | 11 |
| 1276, wax | Mar. 9 | 15 | 2 | 10 | 3 | 0 |
| 1276, no wax | Mar. 9 | 15 | 1 | 0 | 4 | 10 |
| 1247, wax | Mar. 22 | 15 | 0 | 14 | 1 | 0 |
| 1247, no wax | Mar. 22 | 15 | 1 | 0 | 3 | 11 |

From the table can be seen that excellent infection (75%) was obtained from all the inoculations that were protected with wax, while

the checks showed absolutely no infection. In the latter, the wounds had healed over perfectly. The inoculations that were not protected with wax did not give good infection. In the table, the inoculations marked as "doubtful" had dead host tissue around the wound, while those marked "no infection" had the wound entirely healed over. Those marked "infected" had perfectly typical cankers, in many cases with the fungus fruiting. It is possible that some of those marked "doubtful" would have shown typical cankers in one or two months more time. It is interesting to note that with No. 1247, where the first transfer of the culture was used for inoculating, almost perfect infection was obtained.

The technical description of the fungus is as follows:

**Tubercularia Fici** sp. nov.

Sporodochia scattered or gregarious, forming on the surface of the host tissue or underneath a few layers of cells and then becoming erumpent, light pink in color, varying in size up to 3–4 mm. long and 1–1.5 mm. wide. Surface of sporodochia smooth or irregular in outline. Conidiophores crowded, hyaline, 20–27 x 1–2$\mu$, bearing spores at the tips only. Spores small, clear, hyaline, highly refractive, elliptical or oval, regular in size, 5–7 x 2.5–5$\mu$. Setae scattering or abundant, variously placed in the pustules, straight or curved, hyaline or nearly so, septate, papillose, 60–90 x 4–6$\mu$.

Forming cankers around the fruit scars of *Ficus carica*, Baton Rouge, La.

Sporodochiis gregariis vel sparsis, majusculis, superficialibus vel erumpentibus, subroseis. Sporophoris densis, filiformibus, 20–27 x 1–2$\mu$. Conidiis elliptico-oblongis, hyalinis, acrogenis, 5–7 x 2.5–5$\mu$. Setis longis, hyalinis, septatis, papillatis, 60–90 x 4–6$\mu$.

*Hab.* in ramis *Fici caricae*, Baton Rouge, La.

The type material with slides has been deposited in the mycological collection of the Bureau of Plant Industry, Washington, D. C.

THE LIMB BLIGHT OF THE FIG TREE

The second disease of the fig tree, which seems to be new, is one that I have designated as Limb Blight. The disease is characterized by the bright salmon colored fructifications which cover the branches and by the sudden wilting and dying of the leaves on the affected portions (Pl. IV, Fig. 1). Professor E. A. Burt, of Middlebury College, Vermont, has kindly determined the fungus for me, calling it provisionally, *Corticium laetum* Karsten. The fungus generally gains a foothold in the tips of branches that have been killed by the fig canker or by fig borers. While it is a saprophyte at first, it readily grows back on the living branches and out on the living twigs. It

PLATE IV. **Fig Diseases.**

Fig. 1. *Corticium laetum* on fig branches. Shows the fructifications of the fungus and the wilted and dried leaves on the upper branch. Much reduced.

Fig. 2. Cankers on branches of figs, caused by *Tubercularia fici*. Natural size.

causes a death of the cambium layer and consequently causes a wilting and dying of the leaves. The wilting generally takes place at about the same time that the fruiting layer of the fungus forms on the branches. Occasionally, however, the bright colored fruiting layer of the fungus will entirely cover the branch before the cambium is injured enough to cause a wilting of the leaves. In one case noted, the fungus passed out on the petiole of the leaf and on the leaf proper, fruiting in both places, before the leaf wilted.

The fungus, *Corticium laetum*, is not uncommon on dead branches of Alnus in the northern states, but it was not known to be parasitic, nor was it known to occur on fig trees. The fungus fruits very readily forming a bright salmon colored layer over the affected branches. The bright color, however, fades out to a dirty white on old specimens on the trees and also when preserved in the herbarium. In a general way, the fungus resembles *Corticium roseum*, but Professor Burt writes of it as follows:

"I have studied the specimens you sent and believe they are *Corticium laetum* Karsten, rather than *C. roseum*. * * * Thin vertical sections through the fructifications of *C. roseum* will show the fructification composed of two kinds of hyhae, one kind of which is much finer than the other and bears at a certain stage in the life of the fructification minute spherical bodies—perhaps conidia—on short lateral branches. *C. laetum* is composed of only one sort of hyphae and has larger basidiospores than the other. In northern United States and Europe, *C. laetum* has been collected occasionally. It is very bright colored when first found but soon fades. I am very much interested in what you write about your Louisiana specimens."

The fungus also occurs occasionally on apple trees in Louisiana, causing a very similar disease. It has not been observed on other species of trees.

The fungus spreads quite rapidly. In the season of 1908, one tree in the fig orchard on the Experiment Station grounds at Baton Rouge was noticed to be slightly affected, having a few branches killed. In the season of 1909, several of the trees were affected, while in 1910, nearly all of them contained more or less of the disease.

The disease is not a serious one, as it can only gain entrance to the dead tips of the fig branches and does not grow rapidly except during the rainy spells of midsummer. Furthermore only a comparatively small number of the branches of the tree become affected, and as the tree is a very rapid grower, these few are of small consequence.

Specimens of this disease have also been deposited in the mycological collection of the Bureau of Plant Industry, Washington, D. C.

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# FLORET STERILITY OF WHEATS IN THE SOUTHWEST [1]

EDW. C. JOHNSON

For many years in several of the southwestern States particularly in parts of Texas and Oklahoma, wheat yields have been very materially reduced by what has been called floret sterility, due to the non-development of grains in many of the florets of otherwise normal heads. In 1908 careful estimates at San Antonio showed that 30 to 50 per cent of the wheat florets, which ordinarily should develop grains, remained aborted. In 1909 estimates at the same place showed non-development of grains in 12 to 15 per cent of the florets in wheat heads. The trouble was also very noticeable in 1907 but no estimates of the resultant damage were made. Samples sent to the U. S. Department of Agriculture from different parts of Oklahoma and Texas have shown a similar trouble but until recently the causes for the disease have not been accurately determined.

In 1908 investigations of the trouble were begun at San Antonio, Texas. Wheat thrips, rusts and several imperfect fungi were found in the majority of cases where kernels had not developed and work was inaugurated to establish the relation of these agencies to the disease in question.

## INSECTS

In 1908 one or more wheat thrips were found in at least two-thirds of all sterile florets examined. They seemed to be feeding on the stamens and ovaries of the immature and undeveloped grains. In 1909 these insects were also present in large numbers.

A large number of experiments with thrips were performed, such as placing them on wheat heads enclosed in lamp chimneys plugged with cotton at both ends, covering heads with paper bags in which insects were placed, etc. These experiments were not sufficiently complete to allow of definite conclusions.

In the work with insects, however, one point was noticed which is of importance, *i. e.*, the role played by thrips in spore dissemination. Large numbers of insects were examined under the microscope. As many as five rust spores and three conidial spores of imperfect fungi were observed attached to the antennae and appendages of a single thrip. A large per cent of the insects examined were thus seen to be spore carriers. As the thrips are exceedingly active and very numerous large quantities of spores must be distributed in this manner over individual plants and between the different plants in the field. This

[1] Paper read before the American Phytopathological Society at the Boston Meeting Dec. 31, 1909; Abstract appeared in Science, N. S. **31**: 792. 1910.

accounts in part for the prevalence of rust spores between the glumes of florets of the wheat and the consequent rusting of the heads described later in this paper.

### EFFECT ON FLORET STERILITY OF SHADING PLANTS

Thinking that the intense sunlight and heat during clear days might perhaps injure the delicate reproductive mechanisms, a small wheat plat was shaded from the direct rays of the sun before time of heading and during the flowering period. This was done by means of a large piece of paper stretched above the plat on poles thus allowing free circulation of the air and at the same time giving perfect shade. Out of 251 florets from 15 representative heads from this shaded plat 85, or 33.86 per cent, did not develop grains. From 297 control florets, from 15 representative heads picked not two feet from this plat, 64 florets, or 21.54 per cent developed no grains. (Table 1).

TABLE 1.—EFFECT OF SHADING PLANTS FROM THE DIRECT RAYS OF THE SUN DURING HEADING AND FLOWERING PERIODS

| No. of head | Plants shaded from direct rays of the sun | | | Plants adjacent to shaded plants, under natural field conditions. Control | | |
|---|---|---|---|---|---|---|
| | Number | | Per cent sterile florets | Number | | Per cent sterile florets |
| | Fertile florets | Sterile florets | | Fertile florets | Sterile florets | |
| 1 | 8 | 2 | | 11 | 3 | |
| 2 | 14 | 2 | | 16 | 4 | |
| 3 | 8 | 8 | | 15 | 6 | |
| 4 | 4 | 7 | | 16 | 2 | |
| 5 | 9 | 7 | | 20 | 3 | |
| 6 | 9 | 5 | | 13 | 3 | |
| 7 | 7 | 8 | | 12 | 5 | |
| 8 | 9 | 10 | | 13 | 7 | |
| 9 | 14 | 2 | | 21 | 1 | |
| 10 | 17 | 1 | | 21 | 1 | |
| 11 | 13 | 7 | | 20 | 4 | |
| 12 | 13 | 1 | | 28 | 2 | |
| 13 | 14 | 4 | | 9 | 9 | |
| 14 | 8 | 10 | | 12 | 6 | |
| 15 | 14 | 2 | | 6 | 8 | |
| 16 | 5 | 9 | | | | |
| Total | 166 | 85 | 33.86 | 233 | 64 | 21.54 |

This result was unexpected but can be explained by the shaded conditions favoring the development of fungi, the shaded plants remaining moist for a longer time during the morning and after each mist and never drying out as completely as the unshaded plants. The work of these fungi is explained below.

### FUNGI

The ovaries and stamens in sterile florets were attacked almost invariably by one or more fungi. Some of these were *Cladosporium*

*graminum* Cda., one other imperfect fungus, *Stemphylium tritici* Patterson,[1] (a new species here isolated for the first time), and rusts.

*Cladosporium graminum* Cda. At San Antonio *Cladosporium graminum* Cda. is found in great profusion on the leaves of wheat, and, apparently, the same form infects oats and barley. This fungus is very common on diseased ovaries although not always present. Pure cultures were obtained on beef agar with one per cent dextrose, beef agar and oxalic acid agar. On beef agar with dextrose the fungus produced the conidial forms in great profusion. Masses of conidia were placed in sterile water, well mixed and used for inoculation purposes. Normal wheat heads were selected just before and at the time of flowering. The proximal and distal spikelets of the head were cut off and the other florets were inoculated with the fungus by opening the glumes and pouring a drop of water containing conidial spores on each ovary by means of a dropper. The heads were then covered with tissue paper and moistened with sterile water. After two days the paper was removed and the heads allowed to develop under normal conditions. Control heads were treated in exactly the same manner, the glumes being opened, drop of water poured in, covered, moistened, and the cover removed after two days. When the heads were mature examination for undeveloped kernels was made.

Out of 432 florets in 21 heads, inoculated April 17, 135 did not develop grains, thus giving 31.25 per cent sterile florets. In the control, out of 195 florets from 9 heads 55 were sterile, giving a per cent of 28.2 of undeveloped grains. (Table II).

*Stemphylium tritici* Patterson. This fungus was prevalent on the leaves of wheat and almost invariably present in diseased ovaries. Pure cultures were obtained on the same media as those used for *Cladosporium graminum* Cda. Inoculations were made in exactly the same manner as with *Cladosporium*. Out of 186 florets in 10 heads, inoculated on April 17, 56 or 30.10 per cent did not develop grains; while in the control as above, out of 195 florets from 9 heads 28.2 per cent developed no grains. (Table III).

Out of 301 florets in 19 heads, inoculated with *Stemphylium tritici* on April 20, 129 or *42.52* per cent did not develop grains; while out of 198 control florets, similarly treated with sterile water, 67 or 33.33 per cent developed no grains. (Table IV).

*Rusts.* In a very large percentage of sterile florets were found uredospores of rusts, both of *Puccinia graminis tritici* Eriks. & Henn. and *Puccinia rubigo-vera tritici* Carleton. In many such florets the glumes

[1] This species was described by Flora W. Patterson in Bul. Torr. Bot. Club, **37**: 205. 1910.

TABLE II.—INOCULATION EXPERIMENTS WITH CLADOSPORIUM GRAMINUM.

*Inoculations made April 17, 1909, at flowering time of heads.*

| No. of head | Stage of head inoc. * | Glumes opened, water containing conidia dropped on ovary. | | | Stage of head treated* | Control. Glumes opened and sterile water dropped on ovary. | | |
|---|---|---|---|---|---|---|---|---|
| | | Number fertile florets | Number sterile florets | Per cent sterile florets | | Number fertile florets | Number sterile florets | Per cent sterile florets |
| 1 | a | 16 | 6 | | b | 17 | 8 | |
| 2 | a | 10 | 9 | | b | 18 | 4 | |
| 3 | a | 15 | 7 | | a—b | 15 | 7 | |
| 4 | a | 11 | 8 | | a | 12 | 4 | |
| 5 | b | 10 | 11 | | a | 16 | 6 | |
| 6 | b—c | 20 | 2 | | a | 12 | 10 | |
| 7 | b—c | 17 | 7 | | a—b | 15 | 10 | |
| 8 | c+ | 17 | 6 | | a | 16 | 5 | |
| 9 | a | 16 | 6 | | a | 19 | 1 | |
| 10 | a—b | 13 | 11 | | | | | |
| 11 | a | 13 | 4 | | | | | |
| 12 | a | 13 | 5 | | | | | |
| 13 | a—b | 17 | 5 | | | | | |
| 14 | a—b | 16 | 8 | | | | | |
| 15 | a—b | 10 | 12 | | | | | |
| 16 | a—b | 7 | 7 | | | | | |
| 17 | a—b | 11 | 4 | | | | | |
| 18 | a—b | 18 | 3 | | | | | |
| 19 | c | 16 | 6 | | | | | |
| 20 | b—c | 17 | 5 | | | | | |
| 21 | a—b | 14 | 3 | | | | | |
| Total | | 297 | 135 | 31.25 | | 140 | 55 | 28.2 |

* a, Before stamens were mature.
b, At maturity of stamens.
c, Ovary just commencing to enlarge after fertilization.

TABLE III.—INOCULATION EXPERIMENTS WITH STEMPHYLIUM TRITICI

*Inoculations made April 17, 1909, at flowering time of heads*

| No. of head | Stage of head inoc. | Glumes opened, water containing conidia from pure culture dropped on ovary. | | | Stage of head treated* | Control. Glumes opened and sterile water dropped on ovary. | | |
|---|---|---|---|---|---|---|---|---|
| | | Number fertile florets | Number sterile florets | Per cent sterile florets | | Number fertile florets | Number sterile florets | Per cent sterile florets |
| 1 | | 10 | 2 | | b | 17 | 8 | |
| 2 | b | 12 | 5 | | b | 18 | 4 | |
| 3 | b | 18 | 5 | | a—b | 15 | 7 | |
| 4 | b | 19 | 4 | | a | 12 | 4 | |
| 5 | a | 12 | 5 | | a | 16 | 6 | |
| 6 | b | 16 | 4 | | a | 12 | 10 | |
| 7 | a | 11 | 3 | | a—b | 15 | 10 | |
| 8 | a | 7 | 9 | | a | 16 | 5 | |
| 9 | a—b | 11 | 11 | | a | 19 | 1 | |
| 10 | b | 14 | 8 | | | | | |
| Total | | 130 | 56 | 30.1 | | 140 | 55 | 28.2 |

* See footnote, Table II.

TABLE IV.—INOCULATION EXPERIMENTS WITH STEMPHYLIUM TRITICI

*Inoculation made April 20, 1909*

| No. of head | Glumes opened, water containing condia from pure culture dropped on ovary. | | | | Control. Glumes opened and sterile water dropped on ovary. | | | |
|---|---|---|---|---|---|---|---|---|
| | Stage of head inoc.* | Number fertile florets | Number sterile florets | Per cent sterile florets | Stage of head treated * | Number fertile florets | Number sterile florets | Per cent sterile florets |
| 1 | a | 8 | 6 | | a—b | 8 | 6 | |
| 2 | b | 8 | 5 | | a—b | 19 | 3 | |
| 3 | a | 8 | 5 | | | 15 | 6 | |
| 4 | c | 16 | 1 | | a—b | 9 | 7 | |
| 5 | | 11 | 3 | | | 11 | 7 | |
| 6 | b | 5 | 12 | | c | 18 | 6 | |
| 7 | a—b | 9 | 5 | | a | 16 | 0 | |
| 8 | b | 16 | 4 | | | 13 | 9 | |
| 9 | b | 9 | 7 | | a—b | 8 | 10 | |
| 10 | b—c | 12 | 5 | | | 6 | 6 | |
| 11 | b | 9 | 6 | | a—b | 8 | 8 | |
| 12 | | 12 | 12 | | | | | |
| 13 | b—c | 5 | 16 | | | | | |
| 14 | a—b | 8 | 5 | | | | | |
| 15 | b—c | 6 | 6 | | | | | |
| 16 | a | 4 | 11 | | | | | |
| 17 | b | 7 | 5 | | | | | |
| 18 | a—b | 10 | 8 | | | | | |
| 19 | | 9 | 7 | | | | | |
| Total | | 172 | 129 | 42.52 | | 131 | 67 | 33.33 |

*See footnote, Table II.

contained sori of rust on the inside or the outside, or the rachilla and ovary were affected. Often the head was found to be severely rusted while the stem was practically rust free. The rusting of the head in preference to the stem is without doubt due to the fact that the ever present rust spores gain entrance between the glumes either by means of the wind during the flowering season and when the glumes are open in the morning or by means of insects (see above). The spores lodge in the flower more easily than on the stem and are there kept more moist during the greater part of the 24 hours than the spores on the stem, thus having opportunity for germination. In a large number of flowers collected and examined on a foggy morning succeeding a day of scattered showers many spores were found sending out long and vigorous germination tubes, especially between the stigmatic hairs.

To determine whether or not rust spores germinating in the flower can produce sterility, uredospores of *Puccinia graminis tritici*[1] were collected in water and inoculations made on wheat heads just as de-

[1] Some spores of *Puccinia rubigo-vera tritici* necessarily accompanied these as there was no way to secure pure cultures of *Puccinia graminis tritici* in the limited time for the performance of the experiments.

scribed above for *Cladosporium graminum* and *Stemphylium tritici*. In addition, inoculations were made by soaking heads in water full of spores. Control heads were treated in the same manner with sterile water. Out of 93 florets from 7 heads inoculated on April 12 by separating the glumes and pouring water filled with spores into the flower, 48 or 51.61 per cent produced no grains; while out of 206 florets from 12 heads, used for control, 63 or 30.58 per cent produced no grains. (Table V).

TABLE V.—INOCULATION EXPERIMENTS WITH PUCCINIA GRAMINIS TRITICI

*Inoculations made April 12, 1909*

| No. of head | Glumes opened, water containing uredospores dropped on ovary. | | | Control. Glumes opened and sterile water dropped on ovary. | | |
|---|---|---|---|---|---|---|
| | Number fertile florets | Number sterile florets | Per cent sterile florets | Number fertile florets | Number sterile florets | Per cent sterile florets |
| 1 | 6 | 3 | | 11 | 7 | |
| 2 | 13 | 4 | | 24 | 1 | |
| 3 | 6 | 11 | | 11 | 2 | |
| 4 | 5 | 6 | | 7 | 4 | |
| 5 | 6 | 6 | | 13 | 4 | |
| 6 | 2 | 9 | | 13 | 2 | |
| 7 | 7 | 9 | | 10 | 6 | |
| 8 | | | | 3 | 14 | |
| 9 | | | | 6 | 6 | |
| 10 | | | | 16 | 2 | |
| 11 | | | | 20 | 1 | |
| 12 | | | | 9 | 14 | |
| Total | 45 | 48 | 51.61 | 143 | 63 | 30.58 |

Similarly, out of 85 florets from 5 heads, inoculated by soaking heads in water full of spores, 17 florets or 20 per cent developed no grains; while out of 103 florets from 6 heads, used for control, only 13 florets or 12.62 per cent developed no grains. (Table VI).

The last experiment was repeated on April 13, when out of 264 florets from 11 heads 65 or 24.62 per cent did not develop grains; while out of 151 florets from 7 heads used for control 28 or 18.54 per cent developed no grains. (Table VI).

Similar experiments with *Puccinia graminis tritici* were also performed at the Minnesota Experiment Station on winter and spring wheats in 1908. Heads of wheat at different stages of development were selected for inoculations. In 104 florets of winter wheat from 4 heads, inoculated on June 23, 1908, by wetting the heads with water full of spores while the stamens were still green, 10.57 per cent did not develop grains, and out of 119 florets from 5 heads, inoculated when stamens were about mature, 8.4 per cent developed no grains. Control heads treated in the same manner with sterile water gave only 2.94 per cent sterile florets. (Table VII).

TABLE VI.—INOCULATION EXPERIMENTS WITH PUCCINIA GRAMINIS TRITICI

*Inoculations 1 to 6 made April 12, inoculations 7 to 17 made April 13*

| No. of head | Heads soaked in water containing uredospores. | | | | Heads soaked in sterile water. | | | |
|---|---|---|---|---|---|---|---|---|
| | Stage of head inoc.* | Number fertile florets | Number sterile florets | Per cent sterile florets | Stage of head treated* | Number fertile florets | Number sterile florets | Per cent sterile florets |
| 1 | | 18 | 1 | | | 13 | 2 | |
| 2 | | 13 | 4 | | | 15 | 2 | |
| 3 | | 1 | 8 | | | 17 | 3 | |
| 4 | | 18 | 1 | | | 13 | 1 | |
| 5 | | 18 | 3 | | | 14 | 3 | |
| 6 | | | | | | 18 | 2 | |
| Total | | 68 | 17 | 20. | | 93 | 13 | 12.64 |
| 7 | b | 12 | 12 | | | 12 | 8 | |
| 8 | b | 26 | 4 | | | 15 | 5 | |
| 9 | | 19 | 3 | | a—b | 20 | 4 | |
| 10 | | 21 | 3 | | a—b | 14 | 4 | |
| 11 | a—b | 22 | 4 | | b | 19 | 3 | |
| 12 | b | 20 | 4 | | b | 20 | 3 | |
| 13 | b | 19 | 5 | | a—b | 23 | 1 | |
| 14 | a | 22 | 3 | | | | | |
| 15 | a—b | 11 | 8 | | | | | |
| 16 | b—c | 14 | 11 | | | | | |
| 17 | b—c | 13 | 8 | | | | | |
| Total | | 199 | 65 | 24.62 | | 123 | 28 | 18.54 |

*See footnote, Table II.

TABLE VII.—INOCULATION EXPERIMENTS WITH PUCCINIA GRAMINIS TRITICI

*Inoculations on winter wheats at Minnesota, June 23, 1908*

| No. of head | Heads soaked in water containing uredospores. | | | | Heads soaked in sterile water. | | | |
|---|---|---|---|---|---|---|---|---|
| | Stage of head inoc.* | Number fertile florets | Number sterile florets | Per cent sterile florets | Stage of head treated* | Number fertile florets | Number sterile florets | Per cent sterile florets |
| 1 | a | 19 | 6 | | | | | |
| 2 | a | 24 | 2 | | | | | |
| 3 | a | 21 | 1 | | | | | |
| 4 | a | 29 | 2 | | | | | |
| | | 93 | 11 | 10.59 | | | | |
| 5 | b | 18 | 2 | | | | | |
| 6 | b | 29 | 3 | | | | | |
| 7 | b | 23 | 1 | | | | | |
| 8 | b | 15 | 3 | | | | | |
| 9 | b | 24 | 1 | | | | | |
| Total | | 109 | 10 | 8.40 | | 396 | 12 | 2.94 |

*See footnote, Table II.

TABLE VIII.—INOCULATION EXPERIMENTS WITH PUCCINIA GRAMINIS TRITICI

*Inoculation on spring wheats at Minnesota, July 3, 1908*

| No. of head | Glumes opened, water containing uredospores dropped on ovary. | | | | Glumes opened, sterile water dropped on ovary. | | | |
|---|---|---|---|---|---|---|---|---|
| | Stage of head inoc.* | Number fertile florets | Number sterile florets | Per cent sterile florets | Stage of head treated* | Number fertile florets | Number sterile florets | Per cent sterile flore |
| 1 | a—b | 13 | 0 | | a—b | 14 | 2 | |
| 2 | a—b | 24 | 1 | | a—b | 18 | 1 | |
| 3 | a—b | 20 | 2 | | a—b | 20 | 0 | |
| 4 | a—b | 18 | 2 | | a—b | 19 | 2 | |
| 5 | a—b | 11 | 8 | | a—b | 18 | 2 | |
| 6 | a—b | 23 | 0 | | a—b | 13 | 1 | |
| 7 | a—b | 23 | 2 | | a—b | 20 | 1 | |
| 8 | a—b | 18 | 1 | | a—b | 19 | 1 | |
| 9 | a—b | 22 | 1 | | | | | |
| 10 | a—b | 20 | 2 | | | | | |
| Total | | 192 | 19 | 9.00 | | 141 | 10 | 6.62 |

* See footnote, Table II.

From 211 florets of spring wheats from 10 heads, inoculated on July 3, 1908, Table VIII, by pouring water containing spores on the flowers when the stamens were still green, 9 per cent did not develop grains; while from 151 florets from 8 control heads similarly treated with sterile water 6.62 per cent developed no grains.

In the experiments at Minnesota heads just emerging from the boot were also inoculated. At this stage the glumes do not open and water poured on does not easily enter between them. The percentage of sterility was not increased over that in the control by such treatment, except in the case of winter wheat where a slight increase of sterility resulted. The stems and some of the glumes after such treatment rusted badly. It was found in all these experiments that wheat is much more susceptible to stem rust when heading and in the early flowering stage than at any other time in its life history.

The results from rust inoculation were further corroborated at Minnesota in 1909 and 1910. In the plats for rust resistance breeding, all the grains were sprayed with rust spores when in bloom in order to develop a local epidemic, making selection for rust resistance possible. In all the non-resistant wheats, a large per cent of the florets remained undeveloped due to rust infection in the head. Adjacent unsprayed plats showed no such sterility.

## CONCLUSIONS.

The experiments on thrips were not sufficiently extensive for absolute statements as to the relation of these insects to non-development of grains in wheat spikelets, but that they are of importance in the distribution of spores of fungi is strongly indicated.

TABLE IX.—SUMMARY OF INOCULATION EXPERIMENTS

| No. of table | Date of inoc. | Treatment | Number of florets | Per cent sterile florets |
|---|---|---|---|---|
| I | | Plants shaded from sun all day. | 251 | 33.86 |
| | | Control. | 297 | 21.54 |
| II | IV/17 1909 | Inoculated with conidial spores of *Cladosporium graminum*. Glumes opened, spores in water poured on ovary. | 432 | 31.25 |
| | | Control. | 195 | 28.2 |
| III | IV/17 1909 | Inoculated with conidial spores of *Stemphylium tritici*. Glumes opened, spores in water poured on ovary. | 186 | 30.1 |
| | | Control. | 195 | 28.2 |
| IV | IV/20 1909 | Inoculated with conidial spores of *Stemphylium tritici*. Spores in water poured on ovary. | 301 | 42.52 |
| | | Control. | 198 | 33.33 |
| V | IV/12 1909 | Inoculated with uredospores of *Puccinia graminis tritici*. Glumes opened, spores in water poured on ovary. | 93 | 51.61 |
| | | Control. | 206 | 30.58 |
| VI | IV/12 1909 | Inoculated with uredospores of *Puccinia graminis tritici*. Heads soaked in water containing spores. | 85 | 20. |
| | | Control. | 106 | 12.64 |
| | IV/13 1909 | Inoculated with uredospores of *Puccinia graminis tritici*. Heads soaked in water containing spores. | 264 | 24.62 |
| | | Control. | 151 | 18.54 |
| VII | VI/23 1908 | Inoculated with uredospores of *Puccinia graminis tritici* at Minnesota. Heads soaked in water containing spores. | 104 | 10.57 |
| | | | 119 | 8.40 |
| | | Control. | 408 | 2.94 |
| VIII | VII/3 1908 | Inoculated with uredospores of *Puccinia graminis tritici* at Minnesota. Glumes opened, spores in water poured on ovary. | 211 | 9.00 |
| | | Control. | 151 | 6.62 |

The inoculation experiments with *Cladosporium graminum* Cda. tend to show that the effect of this fungus is but secondary and of little or no pathogenic importance as the difference of 3 per cent sterility in florets inoculated and in control florets is very small and can be accounted for by unavoidable differences in heads used for inoculation and for control.

*Stemphylium tritici* Patterson undoubtedly is of pathogenic importance as the difference in percentage of the number of non-developed ovaries between florets inoculated and control florets, though only 2.44 per cent in one experiment, was 9.09 per cent in another where several hundred florets were used for the work.

The experiments with rust show that it is the cause of floret sterility to a large extent. Where inoculations at San Antonio were made directly on the wheat flower there was as much as 21.03 per cent larger number of undeveloped grains in the heads inoculated than in the control heads, and two experiments where inoculations were made by simply wetting the heads with water full of spores

gave, respectively, 7.38 per cent and 6.08 per cent larger number of non-developed kernels in inoculated heads than in control heads. Experiments at Minnesota in 1908, 1909 and 1910 showed that if wheat heads at the proper stage of development are inoculated with rust spores floret sterility will result to some extent even in this region.

Histological and cytological details of these experiments have not been worked out. In the case of *Stemphylium tritici*, however, numerous sections of ovaries and stamens show that the mycelium ramifies throughout the tissues of the ovary and conidial spore forms are produced on the surface. Just how the rust prevents the development of the ovaries is still questionable. In some cases, perhaps, it interferes with normal pollination. In other cases it attacks the ovary directly as seen by the fact that many undeveloped grains have small rust sori on their surfaces. When the rust attacks the ovary early enough it prevents its enlarging, and other semi-parasitic fungi, like *Stemphylium tritici* complete the process of destruction, while if it strikes the ovary a little later, *i. e.*, after it is fertilized and has commenced to enlarge, a rusted kernel often results.

Undoubtedly the most important agents causing floret sterility at San Antonio and vicinity are rusts. Other fungi, chief of which is *Stemphylium tritici*, are associated more or less closely with them.[1]

[1] The experiments of 1909 at San Antonio were conducted during an extremely dry season when there was on an average only about 15 per cent of sterile florets in wheat heads as against 30 to 50 per cent the preceding year. Conditions were thus much more favorable for open field inoculations and observations of results than if the per cent of sterile florets under natural conditions had been much larger.

U. S. DEPARTMENT OF AGRICULTURE,
WASHINGTON, D. C.

# BLACK-LEG OR PHOMA WILT OF CABBAGE

## A NEW TROUBLE TO THE UNITED STATES CAUSED BY PHOMA OLERACEA SACC.[1]

THOS. F. MANNS

(WITH PLATES V AND VI)

A cabbage disease has recently occurred in several districts of Ohio which has not been recorded; this threatens to be a serious trouble. It has been found throughout the Clyde and Fremont districts in Sandusky county, and the Greenspring district in Seneca county, and the losses in some instances have been large. Specimens have also been received from other parts of the state. Since no mention could be found in literature which indicated that this disease has been found previously in America, it is presumed the trouble is somewhat new with us. A similar disease in Australia has been described by McAlpine[2] as "black-leg" or "foot rot" of cabbage. Since the disease in Ohio districts is quite identical with that in Australia, and is caused by a fungus of the genus *Phoma*, the trouble has been described here under the name black-leg or *Phoma* wilt, the latter name being given to distinguish it from the *Fusarium* wilt with which it is associated. The latter trouble has been noted by Harter[3]. Both of these diseases are known in the Clyde and Greenspring cabbage districts. The *Phoma* trouble is locally designated as "wilt", while the latter (the *Fusarium* disease) is called "yellows". In France, Prillieux[4] has designated the *Phoma* trouble "pourriture des pieds de chou", and states that the disease is of considerable importance; it attacks and rots the stems of a cabbage grown for fodder purposes. The disease is especially prevalent in the province of Vendée. In Australia, McAlpine writes of the disease as "perhaps the most serious trouble with which the grower has to contend." No mention is made by McAlpine of the presence in Australia of the "black rot" or of the *Fusarium* "wilt" diseases of cabbage. He assigns as the cause of black-leg the same fungus as noted by Prillieux, viz.—*Phoma Brassicae* Thum.

The disease has been carefully described and experimentally worked

[1] Paper read before the American Phytopathological Society at the Minneapolis meeting, Dec. 29, 1910.

[2] McAlpine, D. Fungus Diseases of Cabbage and Cauliflower in Victoria and their Treatment. (1901). Dept. of Agriculture, Victoria.

[3] Harter, L. L. The Fusarium Wilt of Cabbage. Science, N. S. **30**: 934. 1909.

[4] Prillieux, E. E. Maladies des Plantes Agricoles. **II**: 295–297. 1897.

with both by Bos[1] and Quanjer[2] in Holland, who assign *Phoma oleracea* Sacc. as the active agent in causing the disease. In North Holland the disease in the field is locally known as "fallsucht" (falling sickness). It also causes a storage trouble known as "krebstrünke." Quanjer in his experimental work finds the cabbage fly and maggot to be closely associated in distributing and furthering the disease. Indeed he goes so far as to assert that the control of the "fallsucht" depends chiefly upon the proper control of the cabbage fly. Prillieux and McAlpine do not note insect association as taking part in furthering the disease.

*Symptoms.* The disease may be observed early in the seed beds causing seedlings which have just put out their first true leaves to wilt. Lesions may also be found on the leaves this early. (See Pl. V, Fig. 3). The preliminary symptom is that of white, slightly sunken, elongated oval areas on the stems, usually below the point of leaf attachment. (See Pl. VI, Fig. 3). There appear early in these lesions small black pycnidia equally though somewhat sparingly distributed over the affected areas. Each pycnidium contains myriads of spores which are evidently the source of a rapid dissemination of the disease at the time of transplanting.

In the early stages of the disease the fungus may be plated out in pure culture as the sole occupant of the lesion. Later the lesion breaks and bacterial decay sets in. In the preliminary attack of the seedling or partially matured plant, no leaf discoloration takes place. Finally however as the lesions become confluent and a collar rot is being effected the margin of the leaves take on a reddish tinge; the latter symptom is usually followed by wilt and a quick collapse. (See Pl. V, Fig. 1).

"It is in half to two thirds grown plants that the disease is seen at its worst. Here it causes a rapid destruction of the cambium at a level with the ground, which extends quickly above and below. The fungus penetrates the xylem followed by bacterial decay. The severely affected plants show a metallic bluish-red color on the margins of the outer leaves, with also some evidence of wilt. The rot lesions extend deeply into the stem. As soon as a collar rot is effected the plant collapses with a sudden wilt. Soon the stem becomes so badly rotted that the wind often upsets the plant by breaking its connection with the root, and the plant blows away.[3]"

[1] J. Ritzema Bos, "Krebstrünke und Fallsucht bei den Kohlpflanzen, verursacht von *Phoma oleracea* Sacc.—Zeitschr. f. Pflanzenkr. **16**: 257-276. 1906.

[2] H. M. Quanjer, Neue Krankheiten in Nord-Holland (Drehherzkrankheit, Fall sucht and Krebs) Zeitschr. f. Pflanzenkr. **17**: 259-267. 1907.

[3] Science, N. Ser. **32**: 829-726. 1910.

In the vicinities of Clyde, Fremont and Greensprings the losses have been excessive. In each of these districts the *Phoma* wilt has been associated with the *Fusarium* wilt; the two diseases having almost driven the cabbage growers out of business. (See Pl. V, Fig 1.) Several cases were noted by growers where their fields last year suffered almost total loss from the black-leg. In a field at Fremont put into cabbage both last year and this, the loss from *Phoma* infested plants on date of Aug. 4th of this season had already reached 65%. Very few fields are entirely free from this disease.

*Cause of the disease.* The writer has experienced no difficulty in taking the causative fungus in pure culture from the beginning lesions. The fungus may be plated out before any pycnidia are formed though the latter appear very early in the lesion. Systematic work on the fungus shows it to be identical with that described by Bos and Quanjer in Holland; viz. *Phoma oleracea* Sacc.

*Infection work.* The writer has carried out infection work both with artificial cultures and with washings from infected material. With pure cultures the inoculation was brought about both by incision of the stem at the surface of the ground (100% infection following) and by pouring an abundance of artificially grown spores along rows of seedlings just appearing through the soil. Likewise infection was produced in seedlings by placing artificial cultures with the seed at the time of planting. Seedlings became infected and collapsed before a second true leaf made its appearance, indicating that cabbage maggot association is not essential on the varieties with which the writer was working; viz. All Season and Market Garden. (See Pl. VI, Figs. 2 and 3). The variety All Season is used extensively in the Ohio kraut districts. The infections with original material was even more marked on seedlings than with the artificial material, several of the seedlings collapsing in the seed leaf stage.

The observations made by the writer in the seed beds and fields, together with the infection work upon seedlings of varieties grown here, convince him that the importance of maggot association as noted by Bos and Quanjer in Holland need not apparently hold true for the disease in our districts.

*Control of the disease.* The disease is typically of seed bed origin, although some evidence was met with at Fremont indicating that certain areas of a particular field showed primary soil infection. The disease is carried over from season to season in the infected parts of the cabbage plant. (See Pl. V, Fig. 2).

McAlpine, in the publication already referred to, suggested that a drench of copper sulfate might control the trouble in the seed bed,

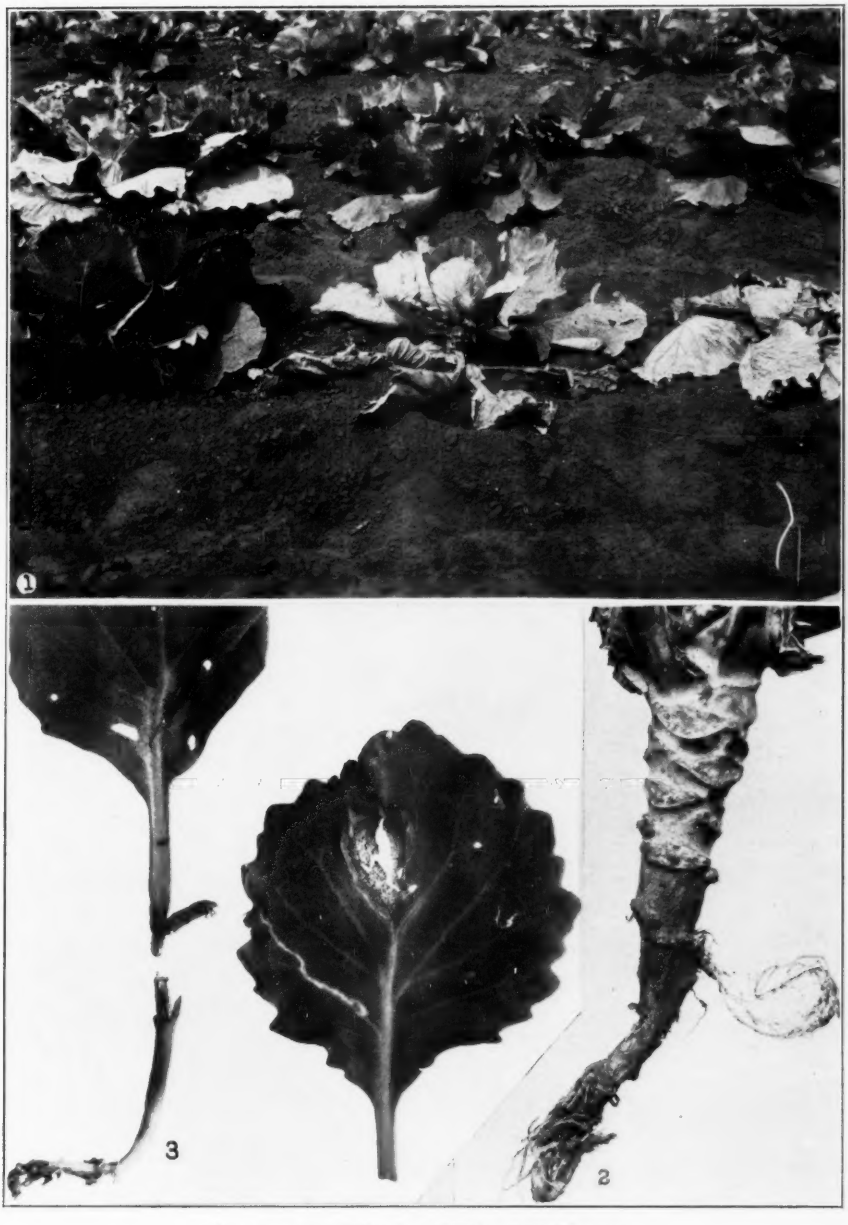


PLATE V. **Phoma Wilt of Cabbage.**

FIG. 1. Contrasting healthy cabbage plant at left with "yellows" sick (*Fusarium* wilt) plant at center, and "black-leg" (*Phoma* wilt) at right.

FIG. 2. Showing the work of "black-leg" on the roots of half matured cabbage plants.

FIG. 3. Showing *Phoma* lesions on cabbage leaves.

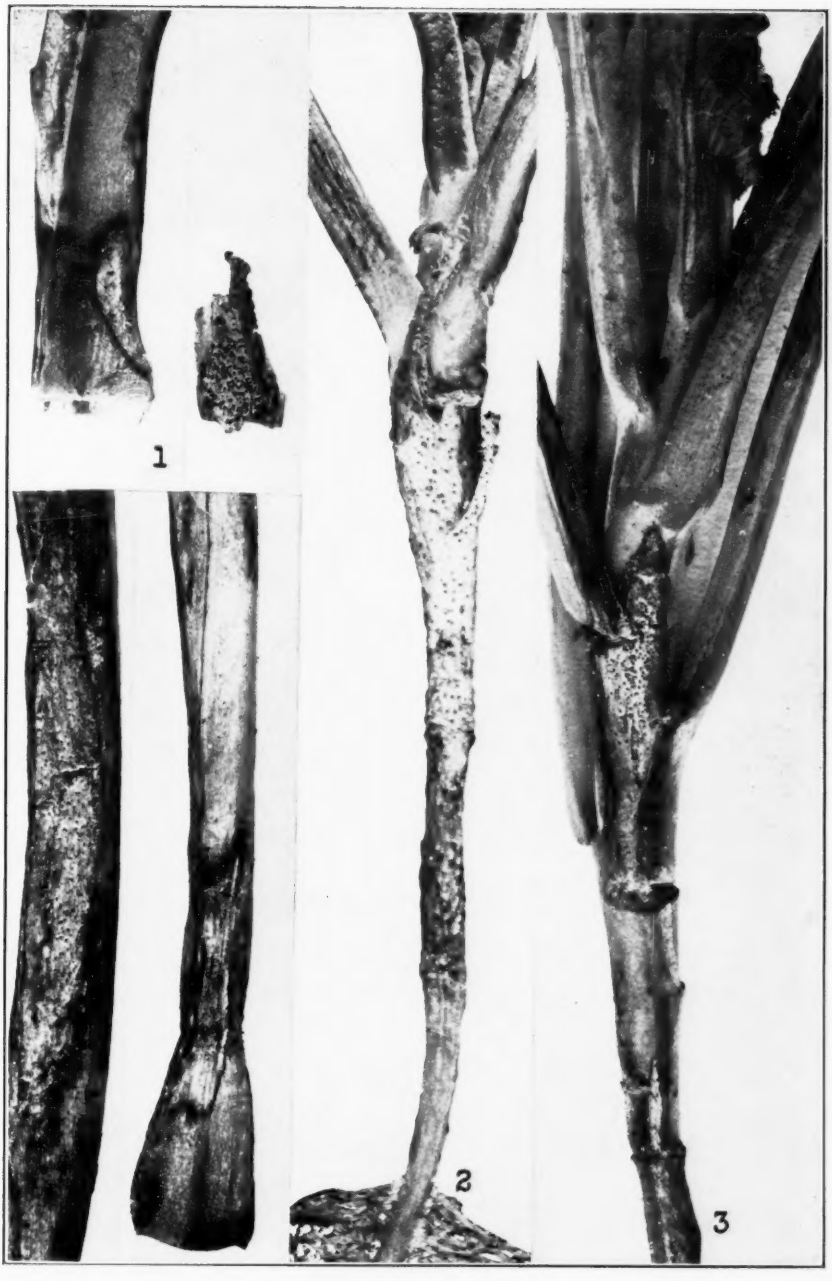


PLATE VI. **Phoma Wilt of Cabbage.**

FIG. 1. Natural, infections, ×2 :—all showing pycindia in the lesions.

FIGS. 2 and 3. Cabbage seedlings infected in the stem and leaf with "black leg" Phoma oleracea Sacc. by placing artifically grown spores of the fungus with the seed at time of planting. Fig 2, ×3 ; Fig. 3, ×2.

since it successfully checked a bed rot of tobacco. His work with copper sulfate on tobacco together with the use of bordeaux mixture in Cuba [1] in the control of a tobacco seed bed trouble and the use by Rolfs[2] in Florida of ammoniacal copper carbonate in the prevention of a tomato disease led the writer to experiment with copper sulfate solution and bordeaux mixture in the control of black-leg in the seed bed. The formulas used were for copper sulfate 4 pounds to 50 gallons of water, and for bordeaux mixture 4 pounds copper sulfate, 4 pounds lime to 50 gallons of water. The solutions were used in two different amounts. The copper sulfate at the rate of 1 gallon to 10 square feet and ½ gallon to 10 square feet. The bordeaux was used at the rate of 2 gallons per 10 square feet and 1 gallon per 10 square feet. The solutions were applied on beds just planted, and on seedlings three weeks old. The copper sulphate solution considerably scorched the leaves of the three weeks' old seedlings though in no way retarded the germinating seed. The bordeaux did not injure the seedlings or retard the germination. Both materials kept down the disease in the seed bed. The bordeaux is being recommended for use in the control of the disease in the seed bed.

Further discussion of this disease, together with other measures for its control, are given in the Ohio Experiment Station Bulletin, No. 225, which is now in press.

OHIO EXPERIMENT STATION,
WOOSTER, OHIO

[1] Horne, Wm. T. Soil Sterilizing, Bordeaux Mixture and Poisons for Tobacco Seed Beds. Esta. Cen. Agron. Cuba. Cir. **30**: 1908; also Cir. **28**: 1907.

[2] P. H. Rolfs. Tomato Diseases—Florida Agr. Exp. Sta. Bul. **91**. 1907.

# A NEW FRUIT SPOT OF APPLE

W. M. Scott

In October, 1907, the writer observed a fruit spot on some Jonathan apples exhibited at the Missouri State Fair, Sedalia, Missouri. From one to a dozen or more spots occurred on each of nearly all the Jonathans on exhibition, representing several different orchards in northern Missouri.

The spots were dark brown in color, circular or somewhat angular in outline, from one-eighth inch or less to one-fourth inch in diameter and were slightly sunken. Although in the main distributed promiscuously over the apple, they were often grouped together on the blush side, which was the upper or sunny side when the fruit was on the trees. In most cases at least, the lenticel formed the center of each spot. The injury was quite superficial, extending into the pulp only slightly.

In the fall of 1908 the same spotting was observed on mature Jonathans that had been lying on the ground several days in an orchard at Barada, Nebraska. Since that time the writer has observed it on Jonathans from Iowa, Missouri and Virginia; on Yellow Newtowns from Virginia, New York and Oregon; on Esopus (Spitzenberg) from Washington and Oregon; and on Grimes (once only) from Washington.

The Jonathan apple, which, until recently, was almost unknown commercially in the Eastern States, is now coming into prominence, especially in the recent plantings. This has brought about a more careful investigation of the variety to determine its weak as well as its strong points. The spotting of the mature fruit has, therefore, become generally known among the newer orchardists as a serious defect of the Jonathan, deterring many from planting it.

The Esopus is perhaps fully as susceptible to this trouble as the Jonathan. During the present fall and winter several carloads of Esopus from the Northwest were marketed in Washington, D. C., and a considerable percentage of them arrived in a spotted condition or developed spots after arrival. On Nov. 18, 1910, a prominent apple grower from the White Salmon Valley, Washington, called at the Bureau of Plant Industry, bringing specimens of spotted Esopus for examination. He had shipped a carload of this variety to New York and upon arrival the fruit was found to be so badly spotted that its market value was reduced as much as fifty cents a box. The writer went to New York to investigate the trouble and on November 21, examined various shipments of apples from the Northwest. The trouble was found on fruit from the Yakima, the Wenatchee and the White

Salmon Valleys, Washington, and to a less extent, from the Rogue River and the Hood River Valleys, Oregon. A box of Esopus apples from one carload was sorted and 34% of the fruit found to be affected. The other lots were not so bad, but there were enough spotted apples in most of the shipments to alarm the dealers.

The cause of the trouble is as yet unknown, but there is a strong suspicion of injury by arsenate of lead used in spraying. In the fall of 1907 Prof. M. B. Waite, of the Bureau of Plant Industry, while in Medford, Oregon, was handed some Jonathan apples affected with this trouble, and was asked to diagnose the case. He expressed the opinion that the spots were probably due to arsenical poisoning. The apples had been picked and kept in common storage for several weeks. Prof. Waite thought that, in sweating, the apples had possibly given off organic acids which dissolved enough of the arsenic to kill the tissues.

In a letter to the writer, dated Nov. 30, 1910, Mr. P. J. O'Gara, until recently, Assistant Pathologist of the Bureau of Plant Industry, states that he has had this trouble under observation for three years, and that in his opinion it is due to the soluble compounds of arsenic in the arsenate of lead used for spraying.

The Bureau of Chemistry has kindly made chemical analyses for us to determine the relative amounts of arsenic in the diseased as compared with the healthy areas. In a letter to the writer, Dr. H. W. Wiley, Chief of that Bureau, makes the following report on these analyses:

"The apples were carefully washed and cleaned in order to remove any arsenic that might have adhered to the skin mechanically. Skin of uniform thickness was then removed from the apples and the arsenic determined in the diseased and normal skin, using the same weight and surface area in each case.

TESTS ON SPOTTED APPLES

| *Arsenic in diseased skin.* | *Arsenic in normal skin.* |
|---|---|
| Test 1—Faint trace[1] | Trace[1] / Faint trace. |
| " 2—0.5 parts per million. | Faint trace. |
| " 3—1.1 " " " | Faint trace. |
| " 4—0.85 " " " | 0.7 parts per million. |

[1] Trace = less than 0.5 parts per million.

"It will be noticed that in most cases the diseased skin contains more arsenic than the normal skin from the same apples; however, in some cases skin from the perfect apples, showing no spots at all, contained as much or more arsenic than was sometimes found in

the spotted portion of the diseased apples. The results indicate a connection between the diseased spots and arsenic content, but we do not consider it conclusive."

The results of these chemical tests support the suspicion of arsenic injury, but as suggested by Dr. Wiley, can not be considered conclusive evidences. The fact that this spotting was apparently not observed until after arsenate of lead came into general use five or six years ago, is rather significant. Moreover, it has not been observed on fruit not sprayed with arsenate of lead, so far as we know, but there has been no systematic investigation to determine this point.

Mr. J. W. Roberts, Scientific Assistant of the Bureau of Plant Industry, and the writer, have undertaken an investigation of this disease and hope to be able to determine its cause by the end of another season. Mr. Roberts has made numerous cultures from the spots, in search of a causal organism, but in most cases no fungi or bacteria have developed. A species of *Alternaria* has been obtained from a number of the spots, but there is no evidence that it is parasitic in this connection.

The only other organism that has occurred in the cultures is a species of *Cylindrosporium*, but this fungus appeared in only a small percentage of the cultures. It resembles Brooks' *Cylindrosporium pomi* which he described [1] as causing a fruit spot of apples in the New England States, and is probably that species. The new spot is very similar to the brown form of Brooks' spot and it seems likely that the two may have occurred together in the same lot of apples, thus accounting for the fungus in the cultures. Moreover, it is possible that the so-called new spot may prove to be the New Hampshire fruit spot, but at present the evidence would not support such a conclusion.

The spots develop mostly, if not entirely, after the fruit is picked and while in temporary or cellar storage, en route to market, or after it is removed from cold storage. Low temperatures retard or prevent its development and the trouble may be avoided in part at least by placing the fruit in cold storage as soon after picking as possible.

U. S. DEPARTMENT OF AGRICULTURE
WASHINGTON, D. C.

[1] Bulletin Torrey Botanical Club **35**: 423-456. Pl. 29-35. 1908.

Diseases of Economic Plants. F. L. Stevens, Ph.D. and J. G. Hall, M.A., 313 pages, 214 figures. The Macmillan Co., New York, 1910. Price $2.00 net.

The appearance within a year of two books on plant diseases, Duggar's Fungous Diseases of Plants and the volume before us, is a noteworthy fact and indicates a new step in the progress of plant pathology in America. Although a vast amount of work in plant pathology has been carried on in this country, and a large number of publications issued, very little effort has been made until recently to collect and present the results of the work in convenient book form for the use of students, investigators and practical agriculturists and horticulturists.

The authors state in the preface that the book is designed to meet the needs of those who wish to recognize and treat diseases without the burden of long study as to their causes, and to indicate the chief prominent characters of the most destructive plant diseases in the United States caused by bacteria or fungi in such a way that in most cases reliable diagnoses may be made. Technical discussions and microscopic details are avoided as far as possible. Though the title of the book does not indicate any restriction as to the classes of diseases discussed, yet we find after a classification of plant diseases into five groups, that only those caused by cryptogamic parasites are treated, with a few exceptions. This restriction in scope is perhaps necessitated in great part by our present lack of knowledge of the causes and treatment of most of the so-called physiological diseases.

A brief historical resumé follows the introduction, indicating the important steps in the development of the subject. The damage caused by plant diseases is next considered, attention being called especially to the great losses caused annually by some of the widespread maladies attacking farm crops and fruits. After a short chapter on symptoms of disease in which the ordinary terms used are defined, methods of prevention and cure are considered. The methods of destroying parasites or preventing their attacks are briefly and concisely stated and should make clear to the practical agriculturist and horticulturist, as well as the student, the manner of infection and the reason for the various methods practised in controlling and preventing disease.

The chapter on fungicides contains a concise account of those in most general use at present with formulas and methods of preparation. Various forms of spraying machinery are briefly described and illustrated and the cost of spraying, especially as applied to fruit, is

discussed, showing the large profits secured by proper treatment. Timeliness and thoroughness in spraying are very properly emphasized, as neglect of one or the other is usually the cause of failure to secure profitable results. Soil disinfection is also briefly treated. A chapter is devoted to general diseases including especially soil and root troubles such as the various root rots.

The body of the work is arranged according to the host plants. The first division includes diseases of special crops. Under this are taken up, first, pomaceous fruits; then drupaceous fruits, followed by small fruits and tropical fruits; next come vegetable and field crops with cereals, forage crops, fibre plants, trees and timber in order, ending with ornamental plants. The different host plants and diseases are arranged in alphabetic order according to their common names, which makes it practicable for the general reader to locate most of the hosts and diseases without difficulty. The description of the diseases, though not technical, give a very good idea of their characters. Many of the half-tone illustrations it is feared will not be of any great assistance to the practical fruit grower or horticulturist who is not already familiar with the disease. This is not due to the character of the reproductions but to the nature of the diagnostic characters of the diseases which are not adapted to this method of illustration.

Under the names of the diseases we find a large number of new terms formed from the generic name of the fungus causing the disease with the termination "ose". Thus for the well-known wilt of cotton we find also "fusariose". It is doubtful whether either the cotton planter or the pathologist will find any great advantage in substituting this name for wilt. The question also might be raised why the name "fusariose" should be used when the organism causing the disease has most frequently been called *Neocosmospora*. This case illustrates very well one of the difficulties of such a system of naming, as the nomenclature of the parasites is in such an unstable condition that many of them are almost certain to be changed, in which case the name instead of helping to remind one of the name of the organism causing the disease would rather lead to confusion. This is, however, not a very serious matter as in most cases where a common name was already in use it is given at the head of the paragraph, thus leaving the choice of names to the reader.

There is an appendix to the book devoted to a few pages of text and illustrations concerning plant physiology and fungi and bacteria, intended, apparently, for the information of practical agriculturists and horticulturists who are not expected to have much special knowledge of these subjects.

The characteristics of the diseases are given in a concise and graphic manner. Nearly all of the fungous diseases of the United States thus far reported are treated, less space being devoted to those of minor importance. From the standpoint of a practical grower the completeness of the work would perhaps rather decrease than increase its usefulness; since in determining a disease the greater the number described the greater in general would be the difficulty of identification.

A few typographical errors, especially those which appear on page 100, are likely to be confusing. In giving the combination treatment for apple diseases, reference is made to methods and sprays mentioned earlier in the volume, but the pages cited are incorrect. Fortunately, however, the methods and sprays can be located by the numbers used. As formulas and directions for spraying are exceedingly important practical matters, a repetition of these would have been amply justified. Bold-faced type beginning the paragraphs relating to the treatment of the diseases would also have been a great convenience to the practical agriculturist and horticulturist in locating quickly this important part of the text.

The references to literature are well selected. They are not numerous, but sufficient for the purposes of the general readers for whom the book is primarily intended. Their value to the specialist might have been increased by more critical editing so as more uniformly to include dates of publication and page citations, and also to eliminate such errors as occur on page 131 where a publication of Smith and Townsend on crown gall is attributed to Hedgcock. The work is a valuable addition to pathological literature. It will, however, we believe, be found more useful to agricultural students and beginners in pathology, than to farmers and fruit growers, while the professional pathologist may frequently find it a convenient work for reference.

C. L. SHEAR.

# EDITORIAL ANNOUNCEMENT

The American Phytopathological Society is entering upon its third year. Its membership, which now amounts to 200 persons, includes most of those professionally engaged in phytopathological research or teaching in the United States and Canada as well as others whose interest is less technical. From the outset it was evident that the Society needed an official organ to provide a place for the publication of many papers which might otherwise be lost or scattered in various places. At the Boston meeting, December, 1909, the Executive Committee was authorized to appoint a Board of Editors and proceed with the organization of plans looking toward such a publication.

Although developments were well advanced several months ago, it seemed to the Editorial Board best to await the opportunity afforded by the Minneapolis meeting of the Society, December, 1910, to present these plans for the final ratification of the Society before beginning the publication. Acting under the further authorization of the Society there given the journal is now started. The Editors wish at the outset to express their appreciation of the kindly interest and the patience shown by the Society in their work, and especially for the helpful suggestions received from many members. The continued success of PHYTOPATHOLOGY depends upon the sympathetic cooperation of the members of the Society as individuals even more than upon the attitude of the Society as an organization.

The plan for the current year is to issue six numbers, generally similar in size and content to this one. The natural expectation will be for enlargement in the future as rapidly as experience shows this to be justifiable. This journal is designed primarily as a channel of publication for the phytopathological contributions of the members of the Society. Much of the space will naturally be occupied by the papers read at the Society's meetings. The editorial policy, however, will aim to make the journal more broadly representative than this and we hope to deserve and receive the subscriptions and support of all who are interested in the study of plant diseases. Its pages will be open at any time to suitable contributions from any source. While papers will be especially welcome which deal in a fairly complete way with special problems, space will also be given to minor articles or brief notes.

The one point which the Editors would again emphasize is that this is to be the journal of the Society as a whole. Each member should feel free to offer suggestions for its improvement and should share in the responsibility for its continued success.